

## REMARKS

Claims 1-2, 5-7, 9, 34-35, 42, 46, 50, 54 and 58-66 are pending. Applicant has cancelled claims 1-2, 5-7, 9, 34-35, 42, 46, 50, 54 and 58-66, without prejudice, and has added new claims 67-109. New claims 67-109 more clearly point out subject matter of the cancelled claims and find support throughout the specification and claims as originally filed as discussed below. No new matter has been entered.

***35 U.S.C. § 103(a)***

Claims 1, 5-7, 9, 34, 35, 50, 54, 59-63, 65 and 66 stand rejected under 35 U.S.C. § 103(a) as being unpatentable over Guo et al. (1994) in view of Sornasse et al. (1992) and Young, et al. (1990).

Applicant has cancelled the instant claims without prejudice, rendering their rejection moot. However, Applicant traverses the rejection as it would apply to newly added claims 67-109. Newly added independent claims 67 and 88 are each drawn to a method of producing a plurality of dendritic cell/tumor cell hybrids capable of inducing a primary anti-tumor response specific to said tumor cell, comprising the step of fusing a tumor cell to a dendritic cell of bone marrow, blood or lymph, and selecting for dendritic cell/tumor cell hybrids which express tumor associated cell antigens of said tumor cell in association with a MHC Class II polypeptide. Dependent claims require that the hybrids further express ICAM, and/or B7.

Below, Applicant explains that a simple substitution of the dendritic cells as taught by Sornasse et al. into the methods of producing fusions of activated B cells to tumor cells, as taught by Guo et al. would not be obvious. That is, in view of the down regulation of the MHC Class II and B7 observed in the B cell hybrids taught by Guo et al., (Figure 1 of Guo et al.), and the lack of knowledge as to the expression of MHC class II and B7 markers on dendritic cell/tumor cell hybrids (see expert declaration, paragraph 14), it would not have been obvious at the time of the invention that a dendritic cell/tumor cell hybrid produced would possess the characteristic of upregulated MHC Class II molecules sufficient to induce a primary response as required by the claims and as disclosed in the FACS staining illustrated Figure 9 of the specification and the in vitro and in vivo assays disclosed Example 12 of the specification.

*Graham v. John Deere Co.*, 338 U.S. 1, 148 USPQ 459 (1966), recently reaffirmed by *KSR International Co. v. Teleflex Inc.*, 127 S.Ct. 1727, 82 USPQ2d 1385 (2007) provides the analytical framework for determining obviousness. Under *Graham*, obviousness is a question of law based on underlying factual inquiries that address (1) the scope and content of the prior art, (2) the differences between the claimed invention and the prior art, and (3) the level of ordinary skill in the pertinent art. Evidence of secondary factors (e.g., commercial success, long-felt but unmet need, and unexpected results) are also given weight in the analysis. Moreover, to establish a prima facie obviousness rejection of a claimed invention, all the claim limitations must be taught or suggested by the prior art. *In re Royka*, 490 F.2d 981, 180 USPQ 580 (CCPA 1974).

Guo et al teaches a method of making a hybrid comprising tumor cells from Wistar rats and B cells from these rats, and using the hybrids as an immunogen in Wistar rats. Specifically, Guo et al. teach a method of fusing hepatocellular carcinoma derived from Wistar cells with activated B cells from Wistar rats to produce a hybrid which acted as an immunogen when injected into Wistar rats.

The Examiner indicates that the Guo reference differs from the claimed invention only in that it does not teach the use of a DC as the antigen presenting component of the hybrid. The position of the Examiner is that one of ordinary skill in the art at the time the invention would have been motivated to substitute a DC for the B cell in said hybrids because “while both B cells and DC are capable of inducing Il-2 secretion in vitro, DCs induce a more vigorous response, including a TH1 response, in vivo, as taught by Sornasse et al.”, page 3 of office action dated April 21, 2009.

The Examiner maintains that DCs can be superior to B cells for antigen presentation.

However, as stated in the attached declaration, **(paragraph 11 of the declaration)** it is not clear that the B cell tumor cell fusions are the actual inducers of the anti-tumor immune response in the rat system described by Guo et al. Figure 1 of Guo et al. reveals that MHC class II expression on the B cell tumor cell fusion is low, as is the expression of the co-stimulatory molecule B7. The declaration notes that the low levels of cell surface expression of MHC class II and B7 are not consistent with the idea that the B cell tumor cell fusions would be able to stimulate induce a primary immune response, which involves the activation of naïve T cells, **(paragraph 13 of the declaration)**. As further noted in the declaration, Guo et al. provides no

in vitro data showing stimulation of naïve T cells by the B cell fusions (**paragraph 13 of the declaration**). It was known at the time that B cell hybridomas don't stimulate naïve T cells.

Thus, one is not convinced that the B cell tumor cell fusions are stimulating the immune system directly(**paragraph 11 of the declaration**). One possible mechanism for the effectiveness of the B cell tumor cell fusions is the B cell component of the fusion provides the fusion with the migratory properties after subcutaneous injection in the rats. Ordinarily tumor hybridoma cells remain in place after subcutaneous injection. By migrating to the lymph nodes, the B cell fusion is able to contact T cells and dendritic cells, and the tumor associated antigens are presented by DC which initiate the anti-tumor immune response (**paragraph 11 of the declaration**).

Because the B cell – tumor cell fusion itself is not initiating the immune response in Guo et al. as described by the declarant in the attached 1.132 declaration, one could not have reliably predicted at the time of the invention that substituting a DC for a B cell in the tumor fusions would have induced an immune response to the tumor, as required by the claims. Further adding to the unpredictability of the proposed substitution is that 15 years after the publication of Guo et al. their results have yet to be replicated in mouse or humans (**paragraph 9 of the declaration**).

At the time of the invention, the development of a therapy that could initiate an in vivo response to tumors was not yet realized. Scientists were experimenting with techniques by which dendritic cells could be used to present tumor associated antigens to the immune system, including pulsing dendritic cells with exogenous antigen, as described by Sornasse et al., and to pulsing dendritic cells with RNA, with limited success, (**paragraphs 5 and 6 of the declaration**). A third technique developed by Applicant of inducing dendritic cells to present tumor associated antigen to the immune system is encompassed by the instantly claimed methods of making dendritic cell tumor cell fusions, (**paragraph 6 of the declaration**).

To Applicant's knowledge, Applicant is the first to have provided the claimed methods of developing dendritic cell/tumor cell fusions capable of the in vivo presentation of tumor associated antigens (**paragraph 7 of the declaration**). Thus, the field of using dendritic cell tumor cell fusions to invoke an anti-tumor response was clearly in its infancy. The newness of the approach of using dendritic tumor cell fusions for tumor immunotherapy negates an obvious to try motivation, according to US case law.

“Second, an invention is not obvious to try where vague prior art does not guide an inventor toward a prior solution. A finding of obviousness would not be obtained where “what was obvious to try” was to explore a new technology or general approach that seemed to be a promising field of experimentation, where the prior art gave only general guidance as to the particular form of the claimed invention or how to achieve it” O’Farrell, 853 F.2d at 903. This expresses the same idea as the KSR requirement that the identified solutions be predictable 550 I.S. at 421; see also Procter & Gamble, see F.3d at 996-97; Kubin, 561 F.3d at 1359-60.”, Bayer Schering Pharma v. Barr Labs. (Fed Cir 8/5/09) , emphasis added.

Applicant’s claimed methods of making dendritic cell/tumor cell fusions effective in tumor therapy that were, at the time of the invention, a promising new field of experimentation, with only general guidance being provided by the art (**paragraph 5 of the declaration**). . Specifically, Applicant’s methods include selecting for dendritic cell/tumor cell hybrids which express tumor associated cell antigens of said tumor cell and are capable of inducing an anti-tumor response specific to the tumor cell, a method not previously reported.

A determination of obviousness must be made based on what a person of ordinary skill in the pertinent art would have known at the time of filing. Neither the teachings of Guo et al. discussed above, nor the teachings of Sornasse et al.’s experiments, provide specific guidance for Applicant’s method of making for dendritic cell/tumor cell hybrids for tumor therapy. Sornasse et al.’s experiments which were designed to develop an immunization procedure avoiding the use of external adjuvant, see abstract, all involve B and dendritic cells which have been exogenously pulsed with antigen before administration to the animal. That is, the B cells and DC cells used by Sornasse et al. have been incubated with external antigen and subsequently administered to the subject. These experiments did not specifically address the ability of dendritic cells to initiate a primary response in vivo by internally processing and presenting antigens on its surface as required by the hybrids of the instant claims.

As discussed above, Figure 1 of Guo et al. illustrates that the cell surface expression of MHC class II and B7 is much lower on the B cell tumor cell fusions than on the activated B lymphocytes, reflecting their likely inability to activate naive T cells in vivo (**paragraph 13 of the declaration**). As further noted in the attached declaration, at the time of the invention, no one knew MHC class II expression would be regulated differently in activated B cells versus dendritic cells (**paragraph 14 of the declaration**). In view of Guo et al.’s teaching that the B cell-tumor cell hybrids express low levels of MHC Class II antigen relative to activated B lymphocytes, one would have predicted at the time of the invention, that MHC Class II

expression in the dendritic cells-tumor cell hybrid would also have decreased relative to dendritic cells (**paragraph 14 of the declaration**). Thus it would not have been obvious at the time of the invention to have substituted a dendritic cell for an activated B cell in the method of making tumor hybrids taught by Guo et al. because at the time of the invention one would have expected that the expression of HLA Class II and B7 would have been down regulated with respect to dendritic cells, rendering them unable to induce a primary anti-tumor response in vivo as required by the instant claims.

Additionally, although not well established at the time of the invention, the processing and presentation of external antigens taught by Sornasse et al. is quite different from the processing and presentation of internal antigens, such as the tumor antigens expressed by the dendritic cell/tumor cell hybrids themselves, as encompassed by the instant claims (**paragraph 16 of the declaration**). Also not well established at the time, is that antigen processing and presentation by B cells differs significantly from that of dendritic cells, regardless of the category of the antigen (**paragraph 16 of the declaration**). Further at the time of the invention, it was not well established that GM-CSF would be effective in upregulating MHC Class II on dendritic cell-tumor cell fusions (**paragraph 15 of the declaration**).

In view of the infancy of the field of tumor immunotherapy at the time of Applicant's invention, the low level of B7 and Class II expression on the B cell tumor cell hybrids taught by Guo et al., and the significant physiological differences between B cells and dendritic cells, it is Applicant's position that it would not have been obvious to one of skill at the time of the invention to have substituted a dendritic cell for a B cell in a method of making the hybrid fusions encompassed by the instant claims.

In view of the claim amendments and comments, Applicant respectfully requests reconsideration and withdrawal of the rejection of the instant claims.

***35 U.S.C. § 103(a)***

Claims 2, 42, and 46 are rejected under 35 U.S.C. § 103(a) as being unpatentable over Guo, et al. (1994) in view of Sornasse, et al. (1992) and Young, et al. (1990) as applied to Claims 1, 5-7, 9, 34, 35, 50, 54, 59-63, 65 and 66 above, and further in view of US Patent No. 5,851,756.

Applicant has cancelled the instant claims without prejudice, rendering their rejection moot. However, Applicant traverses the rejection as it would apply to newly added claims 67-109.

Applicant respectfully traverses for the reasons described above, i.e., that in view of the infancy of the field of tumor immunotherapy at the time of Applicant's invention, the low level of B7 and Class II expression on the B cell tumor cell hybrids taught by Guo et al., and the significant physiological differences between B cells and dendritic cells, it would not have been obvious to one of skill at the time of the invention to have substituted a dendritic cell for a B cell in a method of making the hybrid fusions encompassed by the instant claims.

US Patent No. 5,851,756 was cited by the Examiner to show the use of GM-CSF in inducing DC characteristics. However, US Patent No. 5,851,756's teaching that GM-CSF induces DC characteristics does not provide the required guidance missing at the time of the invention regarding the use of dendritic cell/tumor cell hybrids for tumor therapy. As discussed above, it was not well established that GM-CSF would be effective in upregulating MHC Class II on dendritic cell-tumor cell fusions (**paragraph 15 of the declaration**). Upregulation of Class II and costimulatory molecules such as B7 is distinct from the teachings by the teaching of US Patent No. 5,851,756 that GM-CSF can be used to increase the number of dendritic cells. US Patent No. 5,851,756 does not teach that GM-CSF would be effective in upregulating MHC Class II and/or costimulatory molecules such as B7 on dendritic cell, nor dendritic cell fusions. Without such a teaching, Applicant respectfully submits a prima facie case of obviousness has not been achieved. Reconsideration and withdrawal of the rejection is respectfully requested.

***Rejection under 35 U.S.C. § 103(a)***

Claims 50 and 54 stand rejected under 35 U.S.C. § 103(a) as being unpatentable over Guo, et al. (1994) in view of Sornasse, et al. (1992) and Young, et al. (1990) as applied to Claims 1, 5-7, 9, 34, 35, 50, 54, 59-63 and 66 above, and further in view of US 5,637,483.

Applicant has cancelled the instant claims without prejudice, rendering their rejection moot. However, Applicant traverses the rejection as it would apply to newly added claims 67-109. Applicant respectfully traverses for the reasons described above, i.e., that in view of the infancy of the field of tumor immunotherapy at the time of Applicant's invention, and the significant physiological differences between B cells and dendritic cells, it would not have been obvious to one of skill at the time of the invention to have substituted a dendritic cell for a B cell in a method of making the hybrid fusions encompassed by the instant claims.

U.S. patent 5,637,483 was cited by the Examiner to show the use of irradiation of tumor cells in an anti-tumor vaccine to prevent proliferation of the tumor cells in the patient. Irradiation of tumor cells was known at the time of the claimed invention.

However, US Patent No. 5,637,483's teaching that irradiation of tumor cells in an anti-tumor vaccine to prevent proliferation of the tumor cells upon administration to a patient does not provide the required guidance missing at the time of the invention regarding the use of dendritic cell/tumor cell hybrids for tumor therapy. Since the teaching of the cited patent, either alone or in combination with the other cited references, does not address the infancy of the field of tumor therapy Applicant contends a prima facie case of obviousness has not been achieved. Reconsideration and withdrawal of the rejection is respectfully requested.

***Claims Rejection - 35 U.S.C. 102(b)***

Claims 1, 7, 9, 10, 59, 60 and 66 stand/are rejected under 35 U.S.C. § 102 (b) as being anticipated by Breel et al.

Applicant has cancelled the instant claims without prejudice, rendering their rejection moot. However, Applicant traverses the rejection as it would apply to newly added claims 67-109. Newly added independent claims 65 and 83 are each drawn to a method of producing a plurality of dendritic cell/tumor cell hybrids capable of inducing a primary anti-tumor response specific to said tumor cell, comprising the step of fusing a tumor cell to a dendritic cell of bone marrow, blood or lymph, and selecting for dendritic cell/tumor cell hybrids which express tumor associated cell antigens of said tumor cell in association with a MHC Class II polypeptide. Dependent claims require that the hybrids further express ICAM, and/or B7.

Applicant respectfully traverse the rejection of the instant claims, as applied to newly added claims 67-109, on the grounds that the hybrids taught by Breel et al. do not meet all the limitations of the instantly claimed methods of producing dendritic cell/tumor cell hybrids.

Breel et al. teach the generation of hybrid cell lines in a fully syngeneic setting by fusion of SP2/0 myeloma cells (Balb/C origin) with a lymph node population enriched for DC (also from Balb/C origin), see page 170. The hybrid cells were selected for their expression of the dendritic cell surface NLDC-145 marker. The selection resulted in the generation of four

hybrid cell lines that express the antigen NLDC-145, although they do not show the typical morphology of DC. These hybrids when pulsed with exogenous antigen (KLH), were able to present this nontumor antigen to KLH primed T cells from a syngeneic source (Balb/C mice), see Figure 2 and the Materials and Methods section.

However, the methods of Breel et al. do not include the step of selecting for dendritic cell/tumor cell hybrids which express tumor associated cell antigens of said tumor cell in association with MHC Class II or HLA class II as required by the instant claims as newly amended. In contrast, the hybrid cells taught by Breel et al. were selected for their expression of the dendritic cell surface NLDC-145 marker, as described above.

In light of the above amendments and remarks demonstrating that the methods steps taught by Breel et al. are patentably distinct from the instantly claimed methods, Applicant submits that Breel et al. is not an anticipatory reference. Accordingly, Applicant respectfully requests reconsideration and withdrawal of the instant rejection.

***Claims rejection - 35 U.S.C. § 112, first paragraph – written description/new matter***

Claims 1, 2, 5-7, 9, 34, 35, 42, 46, 50, 54, 59-63, 65, and 66 stand/are rejected under 35 U.S.C. § 112, first paragraph as containing subject matter that was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s) had possession of the claimed invention at the time that the application was filed.

The office action indicates that the specification and claims as originally filed do not provide support for:

- (A) the method of Claim 1 comprising producing a plurality of DC/tumor cell hybrids:
  - a) for “a reduction of the number of tumor cells in a patient”,
  - b) comprising the “allogeneic” DCs of step (a),
  - c) comprising the allogeneic tumor cell characteristic of the same cancer type with respect to said patient” of step (b),
  - d) comprising selecting hybrids “that exhibit DC markers, TAAs and the capacity to activate naive T cells in vitro that can recognize the cancer cells of step (b)”.
- B) The method of claim 9 comprising producing a fused cell product “using PEG” and
- C) The method of claim 34 comprising tumor cells ...sensitive to a drug.



D) The method of claim 59, comprising “a tumor cell line having at least one TAA in common with said tumor sample”.

E) The method of claim 66.

Applicant has cancelled the instant claims rendering their rejection moot. However, Applicant addresses the above rejections to the extent that they apply to the newly added claims. In *Faulkner v Ingals*, 448 F.3d 1357 07 (Fed. Cir. 2006) the court enunciated that examples are not necessary to support the adequacy of a written description, and further that the written description standards may be met even where actual reduction to practice of an invention is absent. Nevertheless, Example 12 of the instant specification provides support for the pending claims as discussed below. An excerpt from Example 12 includes:

“Previous publications have shown that the P1A gene is expressed in P815 mastocytoma and encodes a protein that includes a nonapeptide representing a tumor rejection antigen (P815AB; Brichard et al. (1995); Leth et al. (1995)). Hybrid 38 has been tested for the expression of mRNA specific for P1A and showed that hybrid cells, cultured with or without GM-CSF, as well as P815 tumor cells express mRNA for P1A, whereas DC generated from bone marrow progenitors were negative (FIG. 10). Hybrid 38 is a somatic hybrid (it contains an average of 73 chromosomes) between a dendritic cell, as suggested by the phenotype and function (see below), and a mastocytoma cell, as assessed by expression of mRNA specific for P1A”.

By providing an example of the claimed method for producing hybrids capable of inducing a primary anti-tumor response specific to said tumor cell using dendritic cells isolated from “bone marrow”, and selecting for dendritic cell/tumor cell hybrids which express tumor associated cell antigens of said tumor cell and are capable of inducing an anti-tumor response specific to said tumor cell, i.e. clone 38 described in Example 12 of the instant specification, and thus demonstrating reduction to practice, Applicant has not only met, but exceeded the written description requirements and support for the claims as now pending.

Specifically, Example 12 describes methods for producing a plurality of dendritic cell/tumor cell hybrids capable of inducing a primary anti-tumor response specific to the tumor cell, as recited in claims 67 and 109. The methods of producing the dendritic cell/tumor cell hybrid (clone 38) disclosed in Example 12 comprises a fusion of a tumor cell (from the well established tumor mastocytoma cell line P815) to a dendritic cell of bone marrow, and selecting for dendritic cell/tumor cell hybrid expresses a tumor associated cell antigen (the well established TAA of P815AB) of said tumor cell and a HLA class II (see figure 9 of the

specification), as recited in claim 67. Clone 38 produced by the methods of Example 12 is capable of inducing an in vivo anti-tumor response specific to said tumor cell (see figure 12 of the specification), as recited in dependent claim 68. Figure 13 shows that the hybrids provided mice with an in vivo anti-tumor response that comprises an in vivo induction of immune effectors that contribute to the rejection of said tumor cell in a patient, as recited in claim 72 and which confer resistance to a subsequent challenge with said tumor cell, as recited in claim 71. Figure 12 shows that the hybrids provided mice with an in vivo anti-tumor response that comprises an in vivo induction of immune effectors that reduce the growth of said tumor cell in a patient, as recited in claim 73. Support for the recitation of the limitation of claim 74 that the tumor cell is of a primary culture comprising said tumor cell is found in paragraph 110 of the published application which provides that three types of tumor partners can be prepared: (i) primary cultured tumor cells, (ii) immortal tumor cells, and (iii) drug-sensitive immortal tumor cells.

The use of the HAT sensitive, immortal tumor cell line P815 in Example 12 meets the limitation of claim 75 that the tumor cell is of an immortal cell line, the limitation of claim 76 that the tumor cell is obtained from a drug sensitive immortal cell line, and the limitation of claim 77, that tumor cell comprises at least one tumor associated antigen in common with a tumor of a patient. Example 12 provides that DBA/2 mice were among those used as a source of dendritic cells, and that the P815 tumor is of DBA/2 origin, supporting the recitation of claim 78 that the tumor cell is of a tumor of a patient and wherein said dendritic cell is of said patient. The limitation recited in claim 97 that the tumor cell is of a tumor of a patient and wherein said dendritic cell is of a donor who is a matched HLA compatible donor is found in paragraph 104 of the opublished specification. Example 12 provides for the recitation of claims 80 of the limitation that dendritic cell be isolated by in vitro differentiation of dendritic cell precursors isolated from bone marrow, blood or lymph, and of claim 81, that the in vitro differentiation of dendritic cell precursors be effected through the addition of exogenous cytokines e.g., GM-CSF and/or  $\text{TNF}\alpha$  to the dendritic cell precursors in vitro, by disclosing that bone marrow depleted of lymphocytes, granulocytes and class II positive cells were incubated with 200 ng/ml GM-CSF and 100 U/ml  $\text{TNF}\alpha$ , and cultured for 10 days. The recitation of the limitation of claim 69, 80 and 103-106, that dendritic cell/tumor cell hybrid has been cultured in vitro with GM-CSF and/or  $\text{TNF}\alpha$  and/or interferon is disclosed in Example 12 when describing that “ animals

received 3 or 7 injections of  $2 \times 10^6$  irradiated P815 tumor cells or hybrid cells, cultured or not with GM-CSF, every 5 days starting on day 3 after tumor inoculation”.

Table 2 of Example 10 provides support for the recitation of the limitation of claim 68 that the dendritic cell/tumor cell hybrid expresses ICAM, B7 and class II antigens.

Claims 85-102 basically parallel claim 67-84, except that that claims 85-102 are limited to human. Support for the analogous claims 85-102 is found immediately before the summary section of the specification where it discloses “

The present invention provides DLC or DC/tumor cell hybrids and hybridomas for activating anti-tumor responses. Although the specific procedures and methods described herein are first exemplified using a DBA/2 mouse mastocytoma cell line and DLCs or DCs isolated from syngeneic spleen or from bone marrow progenitors, they are merely illustrative for the practice of the invention. Analogous procedures and techniques are applicable for the treatment of human subjects, as thereafter exemplified using a human osteosarcoma cell line and blood-derived DLCs or DCs. Therefore, DLC or DC/tumor cell hybrids and hybridomas could be used to immunize human patients against their cancer”, paragraph 0095 of the published specification.

The summary section itself contains many preferred embodiments where the dendritic cells and tumor cells used for the fusions express HLA molecules which are human MHC molecules. Further, the definition section of the instant specification refers to human embodiments of the defined terms. For example, when defining dendritic cell surface markers, the specification includes “CD1a for human myeloid dendritic cells”, paragraph 0036 of the published specification.

With respect to written description in the specification for the instantly claimed methods of making a dendritic cell/tumor cell hybrid capable of inducing a primary anti-tumor immune response, Applicant respectfully submits, that despite the lack of working examples for a human dendritic cell/tumor cell fusion, Applicant has not only met the written description requirements and support for the claims as now pending, under *Faulkner*. In light of the above remarks and amendments, Applicants respectfully request reconsideration and withdrawal of the instant rejection.

CONCLUSION

In view of Applicants' amendments to the claims and the foregoing Remarks, it is respectfully submitted that the present application is in condition for allowance. Should the Examiner have any remaining concerns which might prevent the prompt allowance of the application, the Examiner is respectfully invited to contact the undersigned at the telephone number appearing below.

Respectfully submitted,

Date: October 21, 2009

/Amy DeCloux/

Kathleen Williams

Registration No.: 34,380

Amy DeCloux

Registration No.: 54,849

Customer Number 21874

EDWARDS ANGELL PALMER & DODGE

LLP

P.O. Box 55874

Boston, Massachusetts 02205

(617) 239-0451

Attorneys/Agents For Applicant